CLAIMS

1. An inactive Ca^{2+}/calmodulin-dependent protein kinase IIα (CaMKIIα) knockin nonhuman animal, wherein a CaMKIIα gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKIIα is expressed, and thereby a protein kinase activity of the CaMKIIα is specifically impaired while a calmodulin binding capacity of the CaMKIIα and a capacity of multimerizing subunits are maintained.

2. The inactive CaMKIIα knockin nonhuman animal according to claim 1, wherein the inactive CaMKIIα knockin nonhuman animal is produced by a gene targeting method.

3. The inactive CaMKIIα knockin nonhuman animal according to claim 1 or 2, wherein one or a plurality of amino acid residues in a catalytic domain of the CaMKIIα has been modified.

4. The inactive CaMKIIα knockin nonhuman animal according to claim 3, wherein one or a plurality of amino acid residues that is required for binding to ATP has been modified.

5. The inactive CaMKIIα knockin nonhuman animal according to claim 4, wherein a lysine residue that is required for binding to ATP has been modified.

6. The inactive CaMKIIα knockin nonhuman animal according to any one of claims 1 to 5, wherein the inactive CaMKIIα knockin nonhuman animal is a rodent animal.

7. The inactive CaMKIIα knockin nonhuman animal according to claim 6, wherein the inactive CaMKIIα knockin nonhuman animal is a mouse.

8. The inactive CaMKIIα knockin nonhuman animal according to claim 1, wherein brain's nucleus accumbens has lower neuronal activity as compared to that of a wild type.

9. An inactive Ca^{2+}/calmodulin-dependent protein kinase IIα (CaMKIIα) knockin cell, wherein a CaMKIIα gene of one or both of homologous chromosomes is substituted into an inactive type so that an inactive CaMKIIα is expressed, and thereby a protein kinase activity of the CaMKIIα is specifically impaired while a calmodulin-binding capacity of the CaMKIIα and a capacity of multimerizing subunits are maintained.